

Table Baseline sexual characteristics of AIDS cases with and without Kaposi's sarcoma in a cohort of homosexual men in Amsterdam, The Netherlands, 1984-1991

Number of sexual partners	KS- (n=101) Mean (SD)	KS+ (n=26) Mean (SD)
Lifetime*	993 (1000)	1178 (600)†
Last 6 months	28 (29)	21 (14)
Insertive anogenital	10 (15)	11 (12)
Receptive anogenital	10 (11)	9 (15)
Insertive oro-anal (active rimming)	4 (6)	6 (14)
Receptive oro-anal (passive rimming)	8 (15)	7 (10)
Use of nitrite (%)	70	71

* Number of lifetime partners preceding entry; other numbers of sexual partners and nitrite use (ever vs. never use) refer to behaviours during the 6 months preceding entry.

†None of the differences were statistically significant.

men 26 (20.3%) were diagnosed with KS as AIDS defining condition. We compared baseline and subsequently gathered behavioural data among KS and non KS cases during the first 4 years of our study. No significant differences in the number of sexual partners with whom KS cases had practiced active rimming compared with non KS cases were present at any time. Also, no differences were found with respect to any other sexual or related variable. Baseline data regarding a selection of these variables are presented in the table. Among those who ever reported active rimming the percentage diagnosed with KS was almost exactly the same as among those who never reported active rimming (22% vs 21%). Since this and three other cohort studies among homosexual men⁴⁻⁶ have failed to confirm the association between active rimming and the development of KS, the hypothesis needs to be reconsidered. Still, the epidemiology of HIV-related KS suggests a sexually transmitted cofactor in its aetiology.¹

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Primary HIV-1 infection associated with prominent genital ulcers

Genital ulceration is a well-known risk factor for sexual HIV contamination, but has seldom been recorded in primary HIV infections. We report one such case.

A 30 year old heterosexual man, a drug-addict who used sterile material, consulted on the 24 January 1991 for sepsis. He had a 15 day history of tender genital ulcers, pharyngitis for 5 days, a diffuse scaly maculo-pustular rash for 1 day, and complained of general aching with persistent fever (39°C), diarrhoea, nausea, vomiting and odynophagia.

He had a 3 cm ulcer and several smaller ulcers on the foreskin, along the corona of the glans prepuce. They were shallow, soft-based, ragged, whitish and surrounded by a red zone. In addition, round abrasions were scattered over the scrotum (fig). He was also suffering from thrush associated with round superficial ulcerations on the soft palate and inner cheeks. Neither nodes nor spleen were enlarged.

The blood cell counts indicated a pronounced lymphopenia ($0.72 \times 10^9/l$) and a

mild thrombocytopenia. The sedimentation rate was 23 mm at one hour.

The diagnosis of Behcet's disease was excluded.

Blood and skin bacterial cultures were sterile. Tests for *Treponema pallidum* were carried out by dark-field microscopy of genital and oral ulcers, VDRL (Venereal Disease Research



Figure Multiple genital ulcers.

Laboratory) and microhaemagglutination tests. *Haemophilus ducreyi* was assayed by direct examination of swab smears and cultures on enriched media. Specimens from genital and oral ulcers and skin rash were inoculated on human fibroblasts for *Herpes hominis* culture. *Chlamydia trachomatis* cultures were performed on HeLa 229 cells using scrapings of urethral cells. Routine throat, urethral and ulcer cultures were performed. All tests scored negative except for *Candida albicans*, detected in throat culture. Epstein Barr and hepatitis B and C serologies indicated past infections.

ELISA (Abbott Recombinant HIV-1/HIV-2 EIA, Wellcozyme Recombinant) and immunoblot (Dupont Biotech) tests for HIV antibodies were negative. In contrast, a high concentration 4350 ng/l of p24 antigen (HIV AG-1 Abbott) was detected, with 99% reduction by the blocking test.

The patient acknowledged having had unprotected sexual intercourse with a casual female partner at risk for AIDS a fortnight before his genital ulcerations appeared.

His temperature spontaneously fell to normal within 2 days. Mucocutaneous signs and blood cell count defects disappeared within one week.

Two months later, he was symptom-free. The lymphocyte count was normal, and CD-4+ and CD-8+ counts were each $0.930 \times 10^9/l$. He became HIV seroconverted with the whole range of HIV-1 antibodies. Antigen p24 was not detected in serum. No antibodies against either syphilis or *Cytomegalovirus* were produced.

The clinical signs, transient thrush, lymphopenia, a high level of HIV p24 antigenaemia and subsequent seroconversion established the diagnosis of acute HIV infection.¹

Genital ulcers have been known to be one of the primary signs of HIV illness since 1986.²⁻⁵ Our case is characterised by a major p24 antigenaemia.

Biggar² described ulcers on the penis and scrotum of a heterosexual Kenyan man who seroconverted on the 16th day. Gaines³ noted two cases each of ulcers of the penis or anus among 20 HIV seroconverted homosexual men with glandular fever-like illness. Hulsebosch⁴ reported an acute HIV infection associated with ulcers on the corona of the glans penis. Calza⁵ documented an acute HIV infection with scattered painful, non-indurated and clean-based ulcers on the penis and scrotum. A borderline antigenaemia (39 ng/l) was followed by HIV seroconversion. Examination of the genital ulcers by electron microscopy did not reveal viral particles. Rabeneck, however, investigating oesophageal ulcerations related to acute HIV infection in homosexual men, observed retrovirus-like particles by electron microscopy, and detected HIV by culture.⁶

The contaminating virus is likely to cross mucus membranes and result in transient ulcerations. Further investigations are necessary to help understand the process of ulceration.

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